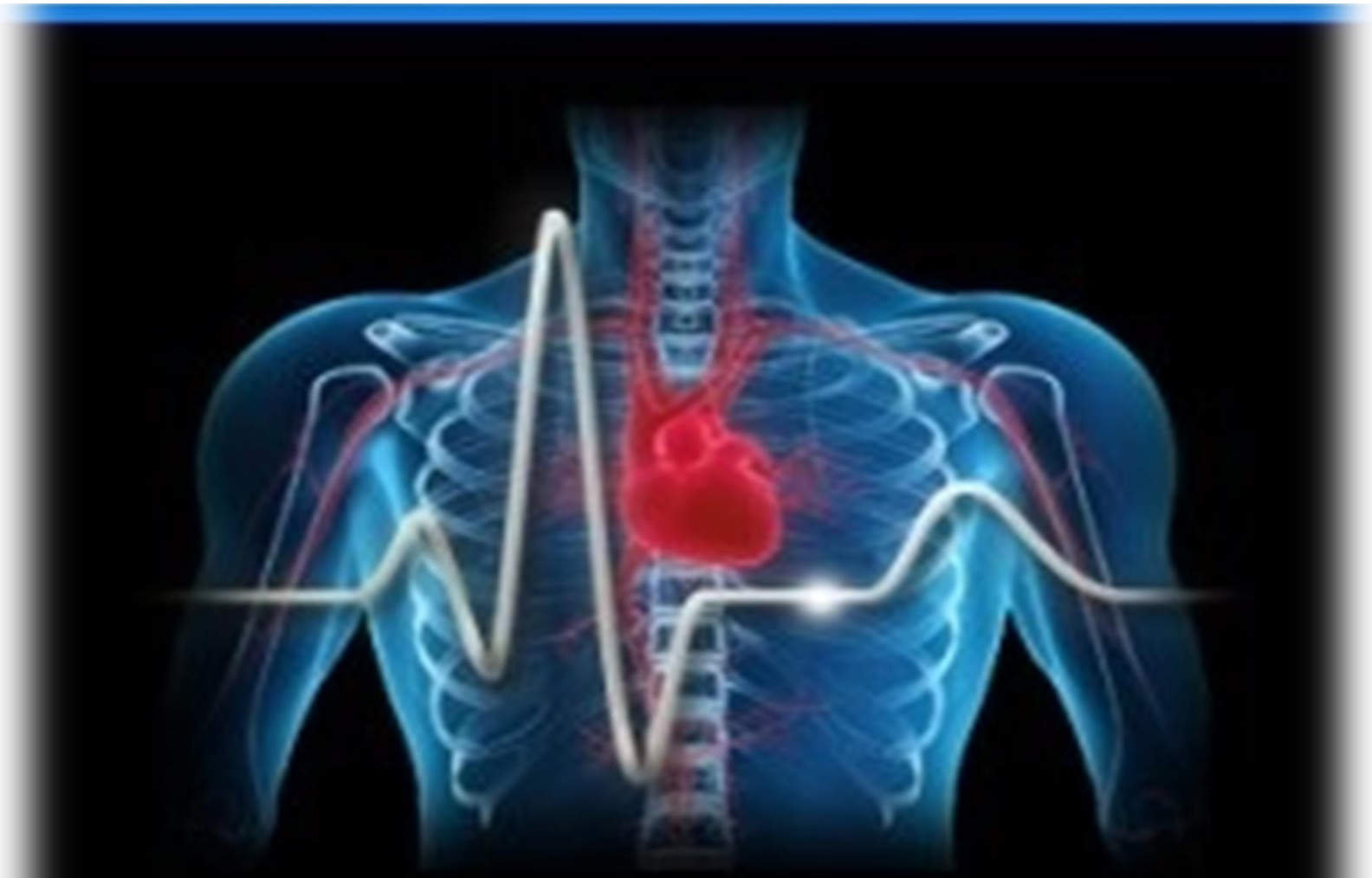
M. Mokhtar Mohammed

ECG Made Easier

ECG made easy ملخص كتاب



إهـــــداء لدفـــعة 2011

إهـــــداء

الحمدُ للهِ جليلِ النعم، باعثِ الهمم، ذي الجودِ والكرم.
الحمد لله ما غرد بلبل وصدح، وما اهتدى قلب وانشرح، وما عم فينا سرور وفرح.

أهدي ثمرة جهدي التي طالما تمنيت إهداءها وتقديمها بأحلى طبق:
إلى التي حملتني وهنا على وهن ، وقاست وتألمت لألمي ، إلى من رعتني بعطفها وحنانها ، إلى أول كلمة نطقت بها شفتاي .. أمي الحبيبية.
إلى الذي عمل وكد وجد فقاس حتى وصلت لهدفي هذا ، إلى من علمني بسلوكه خصالا أعتز بها في حياتي ... فرحمة الله عليم يا أبتِ.

وإلى إخوتي وزملائي، من برفقتهم في دروب الحياة الحلوة والحزينة سرت، إلى من كانوا معي على طريق النجاح والخير. إلى من عرفت كيف أجدهم وعلموني أن لا أضيعهم. وأخُصّ منهم من تعاملت معهم في سنة الامتياز فعلموني معنى الأخوة والعمل الجماعي ومن هم بصدق رسخوا في معتقدي مقولة﴿ معرفة الناس كنوز ﴾وهم عندي أغلى من الكنوز.

وفي النهاية أتوجه بالشكر لله عز وجل الذي أعانني على إتمام هذا لعمل المتواضع وأسأله أن يتقبله مني خالصا لوجهه الكريم. كما لا أنسى أبدا كل من ساهم في إتمام هذا العمل من قريب أو من بعيد أسأل الله أن يثيبهم جميعا خير الثواب. وأخص بالذكر كلا من:
د. أحمد وحيد مصطفى.
د. أميرة مصطفى رشدي.

يا رب لا تدعني أصاب بالغرور إذا نجحت، ولا باليأس إذا فشلت.
يا رب إذا أسأت إلى الناس فأعطني شجاعة الاعتذار، وإذا أساء الناس لي فأعطني شجاعة العفو.

Author
محمد مختار محمد أمين

E.C.G

"Electro – Cardio - Gram"

➢ Electricity of the heart:

- Contraction of muscles =depolarization.

- Although the heart has 4 chambers it can be thought of as having only 2 from electrical point of view.

- Electrical discharge starts at S.A node in Rt. Atrium

➔ spread through A.V node
➔ A.V bundle "bundle of Hiss"
➔ Rt. & Lt. bundle branches

(Lt. bundle divides & spreads through Purkinji fibers)

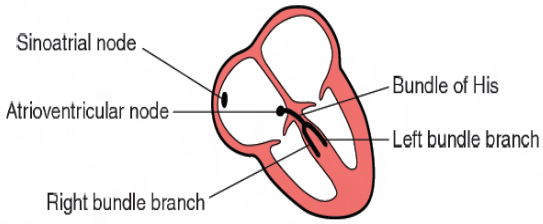
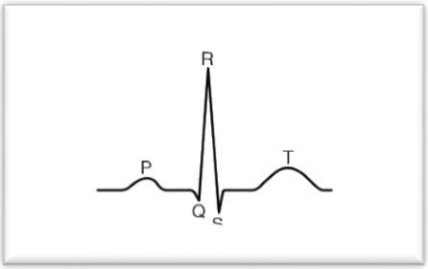


Fig. 1.1 The wiring diagram of the heart

➢ Shape of ECG:



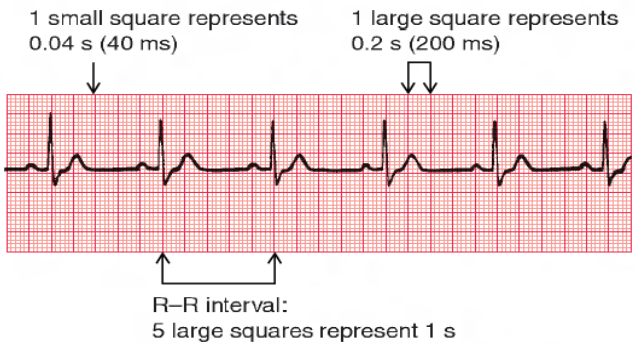
contraction of atria = p wave

contraction of ventricles = QRS complex

relaxation of ventricles = T wave

➢ Time and speed:

- each small square = 1mm = 0.4 sec.
- large square = 5 small squares = 5 x 0.04 = 0.2 sec
- each large square = 0,2 sec
- 5 large squares = 1 sec
- 300 (5 x 60) large squares = 1 min



Heart rate : if regular ➔ 300 / R-R interval (in large squares).

if irregular ➔ no. of QRS in 6 sec. interval. X 10

✓ **P-R interval :**

time taken for excitation wave to spread from SA node through atria & AV bundle to the ventricles ~ 0.12 - 0.2 sec (3-5 small squares)

if decreased > fast conduction

if increased > delayed conduction " heart block"

---

12 leads of ECG

- 6 standard "limb" leads:
  - I , II , III
  - VR, VL, VF

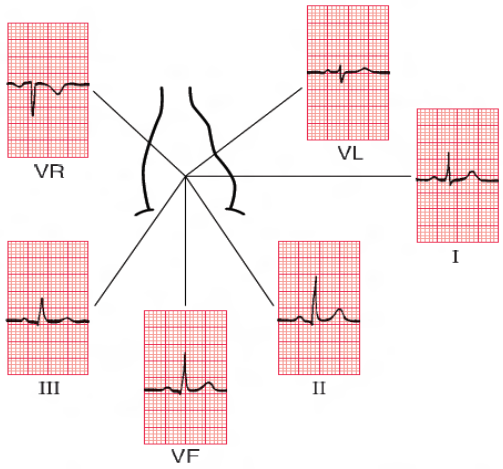


Fig. 1.7 The ECG patterns recorded by the six 'standard' leads

- 6 chest leads:

v1, v2 ➔ Rt. ventricle

v3, v4 ➔ septum

v5, v6 ➔ Lt. ventricle

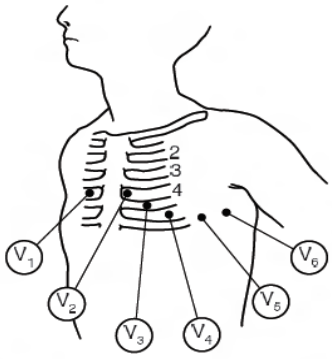


Fig. 1.8 Positioning of the chest V leads. Note that the rib spaces are numbered

In QRS complex:

if QRS is upward > depolarization is moving towards lead

if QRS is downward > depolarization is moving away from the lead

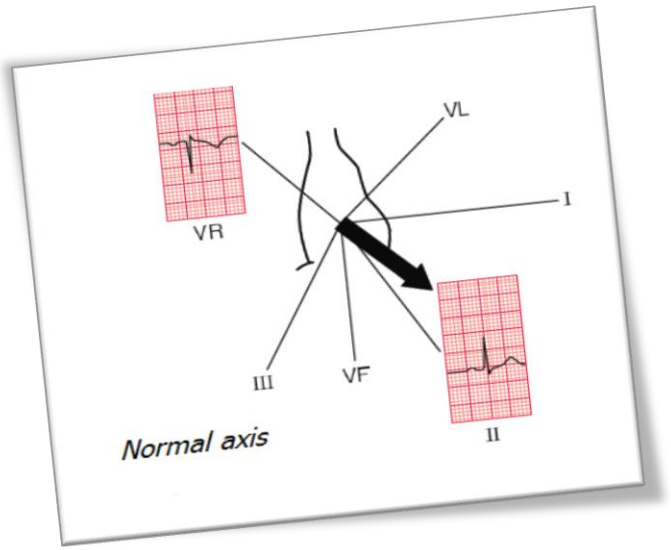
Cardiac axis :

Normal axis: from 11 o'clock to 5 o'clock.
(Moving along VR lead passing by lead II)

Therefore, VR mainly negative "downward QRS" & lead II mainly positive "upward QRS"

In normal axis (11-5 o'clock): Axis is moving toward leads 1, 2, 3

So, 1, 2, 3 are positive (upward QRS).



- (Rt. Axis deviation): axis will swing towards the right.

  - Lead III becomes more positive
  - Lead I becomes more negative
  - as in the case of pulmonary disease & congenital heart diseases. i.e. Rt. Ventricular hypertrophy.

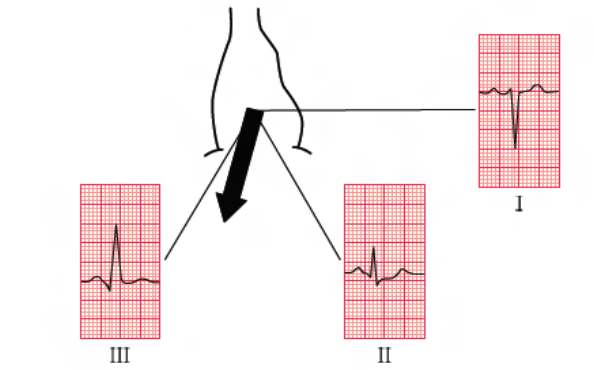


Fig. 1.15 Right axis deviation

- (Lt. Axis deviation): axis will swing towards the left. E.g.: left ventricle is hypertrophy.

  - 3 becomes negative
  - 1 becomes more positive

N.B:

- Lt. axis deviation is not significant until lead 2 become negative. & the problem is usually due to conduction defect rather than increased lt ventricular mass.
- minor degrees of Rt. &Lt. axis deviation may occur in long,thin or short,fat individuals.

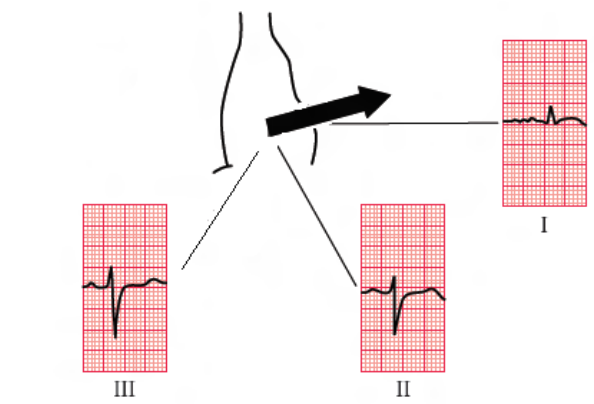


Fig. 1.16 Left axis deviation

QRS complex in V leads:

QRS complex in chest leads shows a progression from V1 (where it is predominantly downward) to lead V6 (where it is predominantly upward).

- Transition point:

- Point where R &S waves are equal.

- It indicates position of septum.

- Normally, between V3 & V4 leads.

- In Rt. ventricular hypertrophy, it becomes between v4&v5 or even between v5& v6.

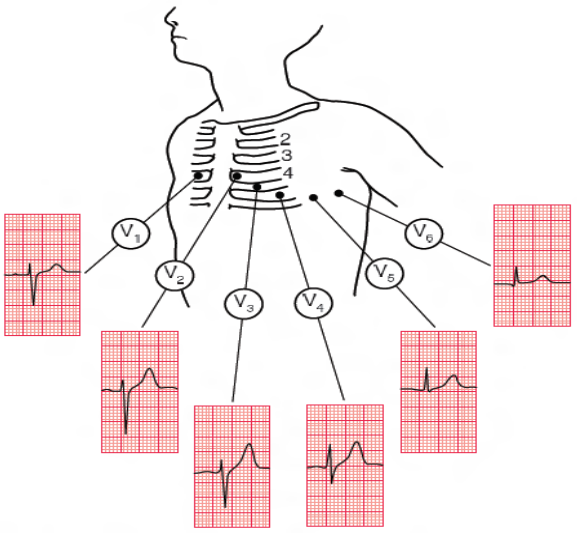


Fig. 1.10 The ECG patterns recorded by the V leads

- ***Comment on ECG :***

1- **R**ate

2- **R**hythm

3- **P**-wave size.

4- **P**-R interval

5- **Q**RS shape

6- **Q**RS duration.

7- ST segment & T wave.

8- Cardiac axis.

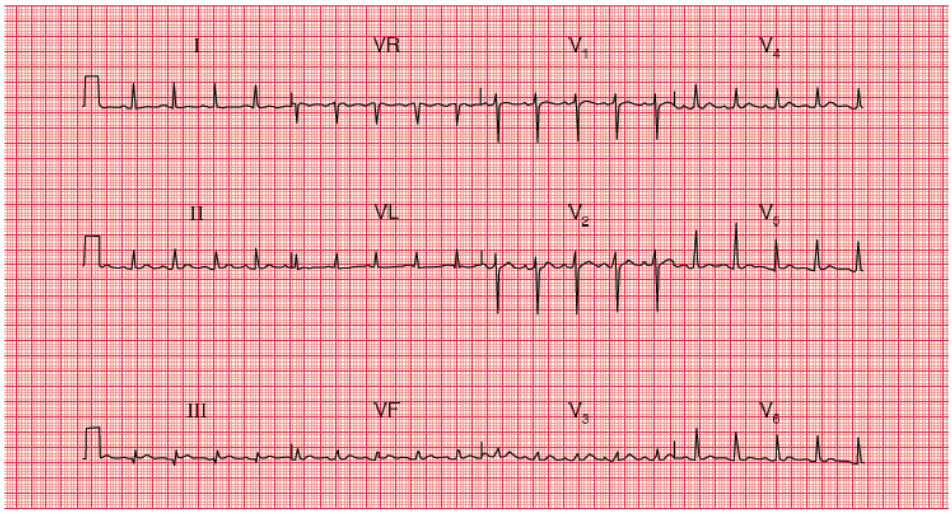


Fig. 1.23 12-lead ECG: example 1

Description

- Sinus rhythm, rate 110/min
- Normal PR interval (140 ms)
- Normal QRS duration (120 ms)
- Normal cardiac axis
- Normal QRS complexes
- Normal T waves (an inverted T wave in lead VR is normal)

Interpretation

- Normal ECG

Conduction and its problems

Normally, conduction spreads from SA node > AV node > AV bundle > Rt. & Lt. bundle branches.

Rhythm of the heart is best interpreted from ECG leads that shows P wave most clearly (usually V1 or lead II)

1-conduction problems in AV node & AV bundle:

PR interval: time taken for excitation wave to spread from SA node through atria & AV bundle to the ventricles ~ 0.13-0.2 sec. (3-5 small squares).

if decreased > fast conduction

if increased > delayed conduction " heart block" (& that's our topic)

✓ 1st degree heart block:

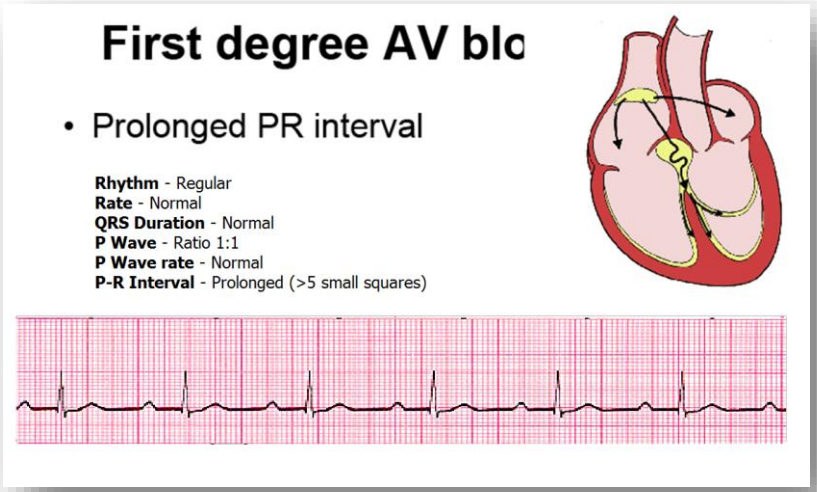
All waves originate in SA node and conducted to the ventricles "normal" . However, there is a delay somewhere along the conduction pathway,

So, PR interval is prolonged. However, is relatively constant from beat to beat.

- Importance of prolonged PR interval:

It may be a sign of :

1. coronary heart disease "CHD"
2. Acute rheumatic carditis.
3. Digoxin toxicity.
4. Electrolyte disturbance.



✓ **2nd degree heart block (intermittent block of conduction):**

Types:

1-mobitz type 2:

(RARE but SERIOUS)

Most beats are conducted normally with constant PR interval, but occasionally there is atrial contraction without subsequent ventricular contraction.

Rhythm - Regular
Rate - Normal or Slow
QRS Duration - Prolonged
P Wave - Ratio 2:1, 3:1
P Wave rate - Normal but faster than QRS rate
P-R Interval - Normal or prolonged but constant

2-wennkebach phenomenon:

progressive lengthening of pr interval & then failure of conduction of 1 beat followed by a conducted beat with short PR interval, then progressive lengthening & repetition of the cycle.

Rhythm - Regularly irregular
Rate - Normal or Slow
QRS Duration - Normal
P Wave - Ratio 1:1 for 2,3 or 4 cycles then 1:0.
P Wave rate - Normal but faster than QRS rate
P-R Interval - Progressive lengthening of P-R interval until a QRS complex is dropped

3- 2:1 type or 3:1 type:

alternate conducted & non conducted atrial beats.

2:1 ➔ one conducted then 2 non conducted beats = 2 p waves per each QRS.

3:1 ➔ one conducted then 3 non conducted beats, the P wave may appear in ECG as distortion of T wave.

2:1 type:
- one conducted then 2 non conducted beats
- 2 P waves per each QRS.

importance of 2nd degree heart block:

it may progress to 3rd degree or it may be a sign of ;

1- CHD
2- Acute rheumatic carditis
3- Digoxin toxicity
4- electrolyte disturbance

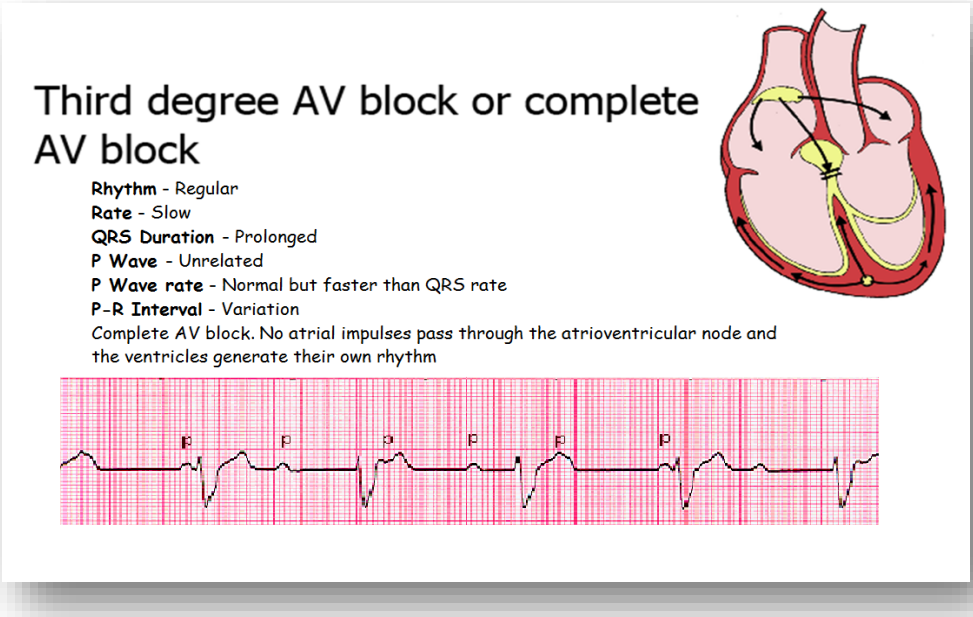
✓ 3rd degree heart block (complete heart block):

Complete block of conduction (no relation between p wave and QRS), mostly due a problem in AV bundle.

It may be:

- Acute as in MI
- Chronic as in fibrosis around AV bundle.

N.B: a pacemaker is essential in treatment.



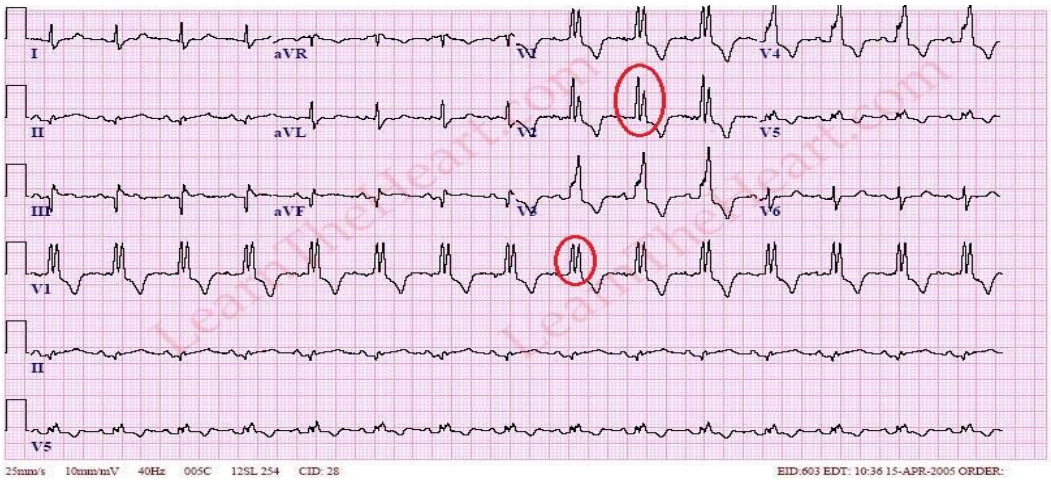
2) conduction problems in Rt. and Lt. bundle branches "BBB" :

In this case the PR interval is normal as conduction pathway is normal, but the QRS complex is wide, normally QRS =120 ms (0,12 sec) (3 small squares), if QRS is wider than 3 small squares > wide BBB > abnormal and indicate that conduction within the ventricles must have occurred by abnormal therefore slow pathway.

- Block of both Rt. & Lt. Bundle= block of AV bundle = 3rd degree heart block.
- RBBB often indicates problem in Rt. side e.g. ASD (but RBBB with QRS complex of normal duration are normal & common).
- LBBB always indicates heart disease (usually left side) e.g. AS and ischemic disease.

RBBB ➔ V1: RSR pattern with wide R-wave **OR** large notched R-wave.

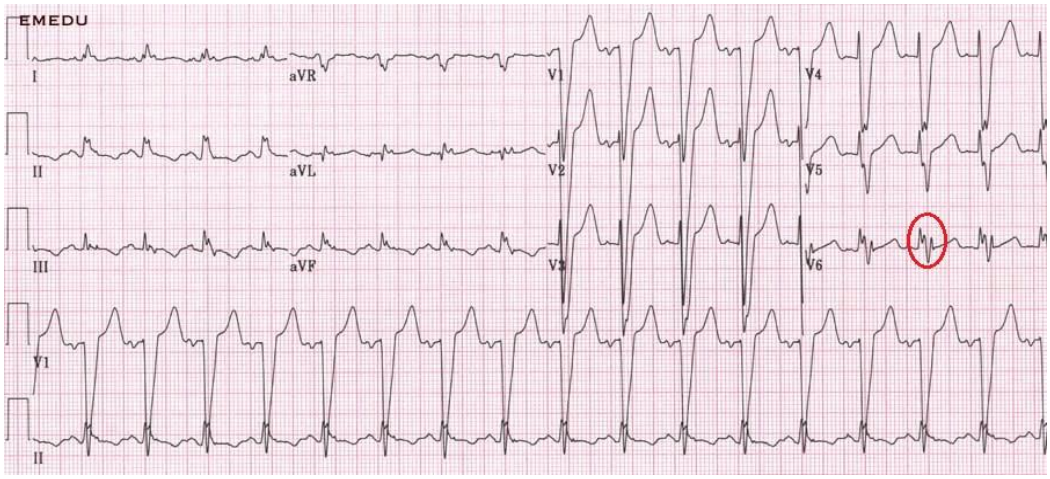
V6: QRS with wide S-wave "pregnant S"



T-wave inversion in Rt. Chest leads are characteristic finding with RBBB

LBBB ➔ V1 : W pattern (wide ,entirely negative QRS complex)

V6 : M pattern (tall wide notched R-wave without Q-wave).



T-wave inversion in Lt. Chest leads are characteristic finding with LBBB

Rhythm of the heart

➢ intrinsic rhythmicity of the heart:

Normally depolarization begin is SA node (highest frequency of discharge) and called "Sinus rhythm".
It may also begin in other places and "Arrhythmia" is said to be present.
The rate of discharge of SA node is influenced by Vagus nerve & reflexes in the lung.

Sinus rhythm:

- Rate = 60 – 100 beat/ min.
- P-wave is positive in lead II
- P-wave is negative in aVR

1. sinus tachycardia:

it's the **most common** arrythmia seen with acute pulmonary embolism.

1-excercise
2- fear
3- pain
4-thyrotoxicosis
5-haemorrhage

Sinus tachycardia : rate is >100 beats/min.

2. sinus bradycardia

1- athletic training
2- fainting attacks
3- hypothermia
4- myxedema
5- hyperkalemia
6- may occurs after heart attacks

Sinus bradycardia : rate is < 60 beats/min.

3. Sinus arrhythmia:

The normal increase in heart rate that occurs during inspiration. This is a natural response and is more accentuated in children than adults.

4. Sinus Pauses, Arrest:

If prolonged ➔ fatal asystole.
Unless : 1. Normal sinus resume.
2. or escape beats from other foci take over.

Escape rhythm :

Normally, rhythm starts in SA node (the most frequent discharge) .

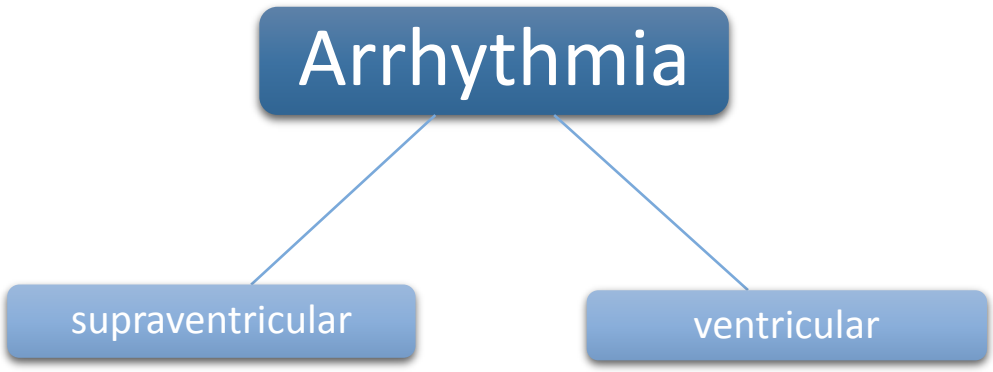
If SA node fail to depolarize ➔ atrial 70 beats/min or AV node 50 /min.

If these fail or BBB ➔ ventricular 30 beats/ min

- This protective mechanism is called **"escape rhythm"** occurs when secondary sites for initiating depolarization escape from their normal inhibition by the more active SA node.
- Escape rhythm is not a primary disorder, but a response to problems in higher conduction pathway.
- They are commonly seen in acute phase of heart attack.
- It is important NOT to try to suppress it because without it the heart might stop.
- The ectopic impulse is always late, appearing only after the next anticipated sinus beat fails to materialize

Abnormal cardiac arrhythmia can begin in:

- Atrial muscle > heart rate = 70 beat /min
- Av node > heart rate = 50 beat /min
- Ventricular muscles > heart rate = 30 beat /min
- may be any where within the atrial or ventricular muscles



1. Atrial premature beats.
2. Paroxysmal supraventricular tachy.
3. Junctional arrhythmias.
4. Atrial flutter.
5. Atrial fibrillation.

1. Ventricular premature beats.
2. Ventricular tachycardia.
3. Ventricular fibrillation.

Supraventricular Arrhythmias

1) Atrial premature beats "APB":

Ectopic stimuli in Rt. Or Lt. atrium (not SA node)

Not conducted to ventricles

- Premature P-wave.
- No QRS.
- Pause.
- Then next normal beats.

Conducted to ventricles
(Atrial Extrasystole)

- Premature P-wave of different shape.
- PR may be different "short or long"
- QRS : no changes.
- Then slight Pause.
- Then next normal beats.

Atrial Extrasystole

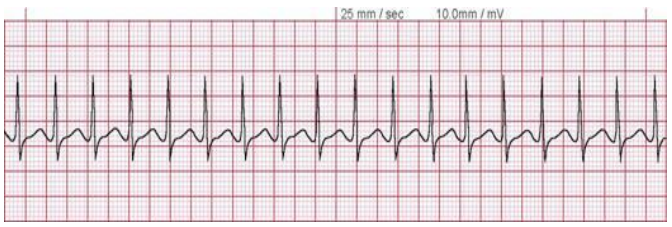
2) Paroxysmal Supraventricular tachycardia:

Definition: Sudden run of 3 or more of premature supraventricular beats.
Types:

a) Atrial tachycardia (3 or more consecutive atrial premature beats).
b) Atrioventricular nodal reentrant tachycardia (AVNRT).
c) Atrioventricular reentrant tachycardia (AVRT).

Atrial tachycardia:

- Rate: more than 150 beats/min
- normal P wave
- normal QRS



Atrial Tachycardia

3) Junctional arrhythmias:

AV junctional rhythm have the following features :

1. P-wave is -ve in lead II and +ve in aVR (retrograde p waves).
2. Retrograde P-wave may immediately precede or follow QRS.
3. In many cases, retrograde p is buried within QRS. When this occurs the baseline between QRS complexes remains completely flat

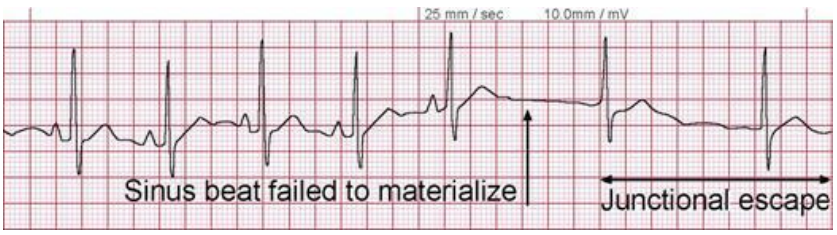
AV JUNCTIONAL RYTHMS ARE CATEGORIZED AS:

a) JUNCTIONAL ESCAPE RHYTM.
b) Junctional tachycardia.

a) JUNCTIONAL (Nodal) ESCAPE RHYTM:

AV node takes over control

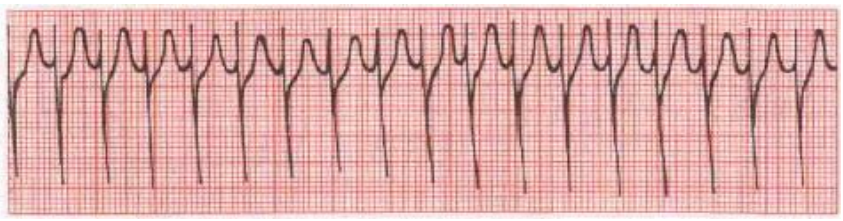
- decreased rate (50/min)
- no p wave
- normal QRS
- may occur singly



b) Junctional (Nodal) tachycardia:

AV junctional tachycardia by contrast, is more rapid run of 3 or more consecutive beats originating in AV junction

- increased rate
- no P wave
- treatment : CSP, &adenosine (if needed)

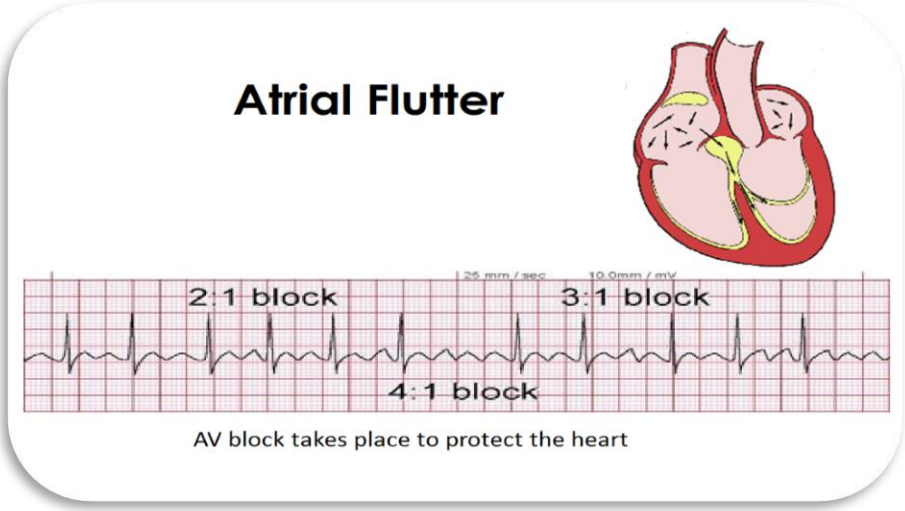


N.B:

- Carotid sinus pressure (CSP): it may be a useful therapeutic effect on supraventricular tachycardia and is always worth trying because it may make arrhythmia more obvious.
- CSP ➔ increase vagal stimulation of SA & AV node ➔ inhibit conduction.
- CSP slows ventricular rate in some supraventricular tachycardia and treats others. but has no effect on ventricular tachycardia.

4) Atrial flutter:

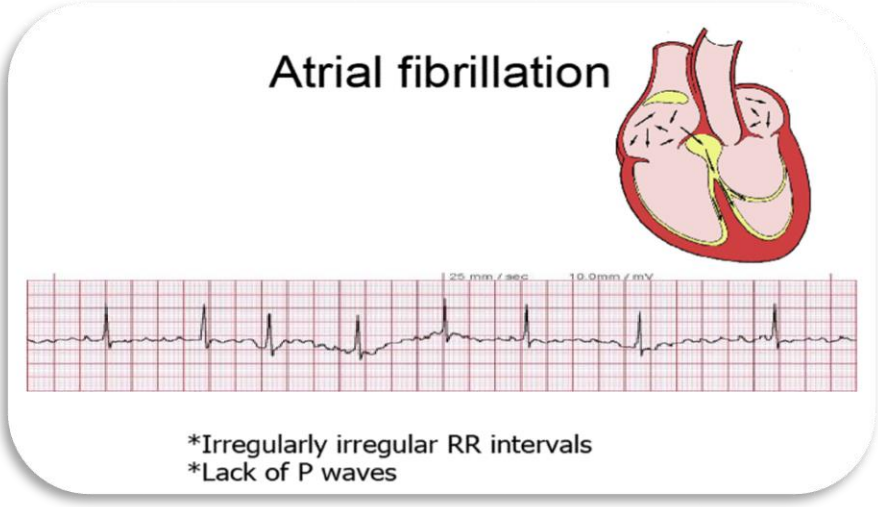
- Atrial rate: more than 250 beats/min
- AV block takes place to protect the heart➔ ventricular rate: 150, 100 or 75.
- Saw teeth appearance (no P-waves).



5) Atrial fibrillation (irregular irregularity):

Def.: non-synchronous contraction of atrial muscle fibers.

No P wave (only irregular baseline), may be flutter like for 2-3 sec.

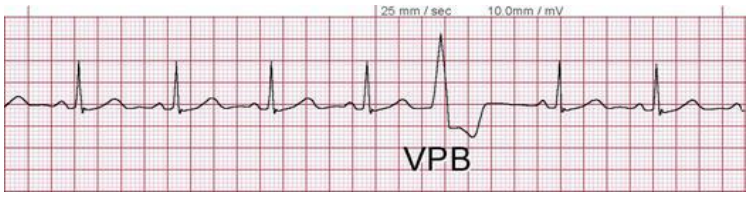


Ventricular Arrhythmias

1) Ventricular premature beat (extrasystole):

3 major characteristics:

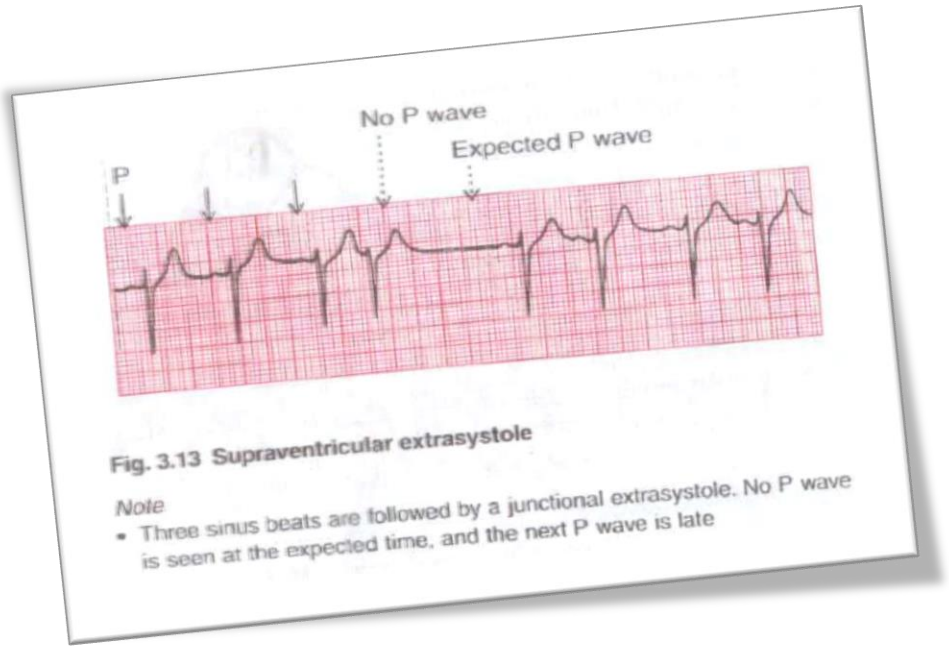
1. Premature beat occurs before the next normal beat is expected.
2. Wide QRS and abnormal T-wave.
3. Followed by a pause.



N.B: effect of extrasystole on following P wave:

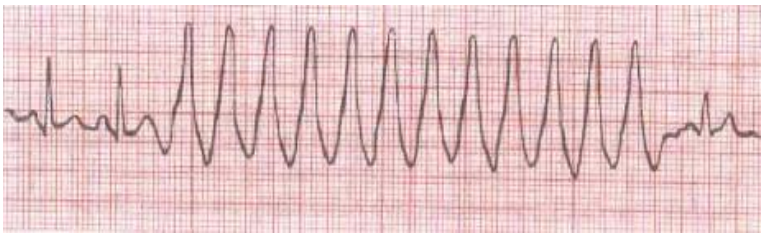
- ✓ Supraventricular ➔ P wave comes late (not in expected time).
- ✓ Ventricular ➔ P wave comes in expected time.

because supraventricular extrasystole is affected by the normal periodicity of the SA node, while ventricular is unaffected.



2) Ventricular tachycardia:

- repeated ventricular extrasystole
- wide abnormal QRS (difference bet it and BBB)
- seen in all 12 leads

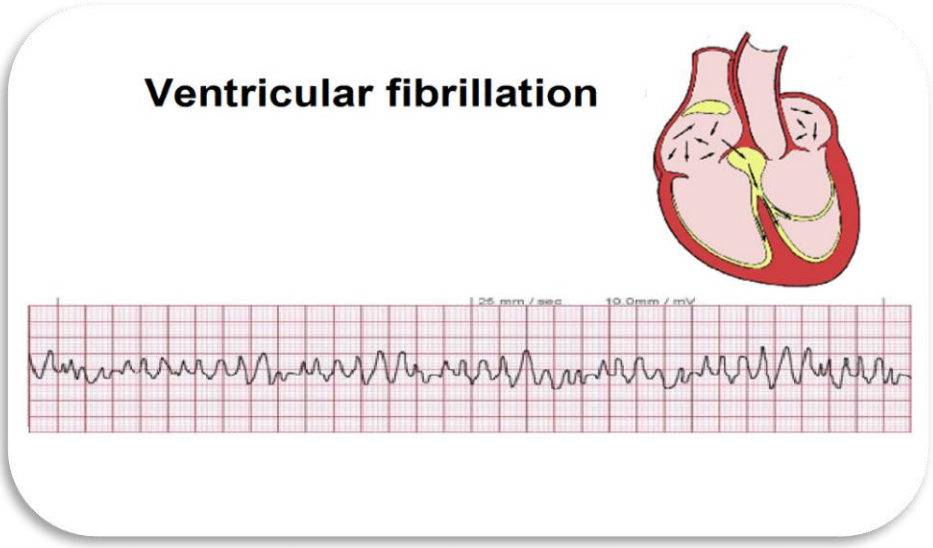


Life threatening condition !!!

3) Ventricular fibrillation :

Def.: non-synchronous contraction of ventricular muscle fibers.

- no QRS identified.
- ECG totally disorganized.
- patient usually is unconscious.



Wolf-Parkinson-White syndrome

- The normal connection between atria and ventricles is the bundle of Hiss.

- Some people have an extra (accessory) pathway, making direct connection.

- Usually, it is in left side of the heart.

- There is no AV node to delay conduction. So, depolarization wave reaches the ventricles early and pre-excitation occurs.

Clinical importance:

It can cause paroxysmal tachycardia. Depolarization can spread down Hiss bundle and back up the accessory path ➔ re activate the atria ➔ sustained tachycardia.

*PR interval is short.

*QRS shows early slurred upstroke "delta wave".

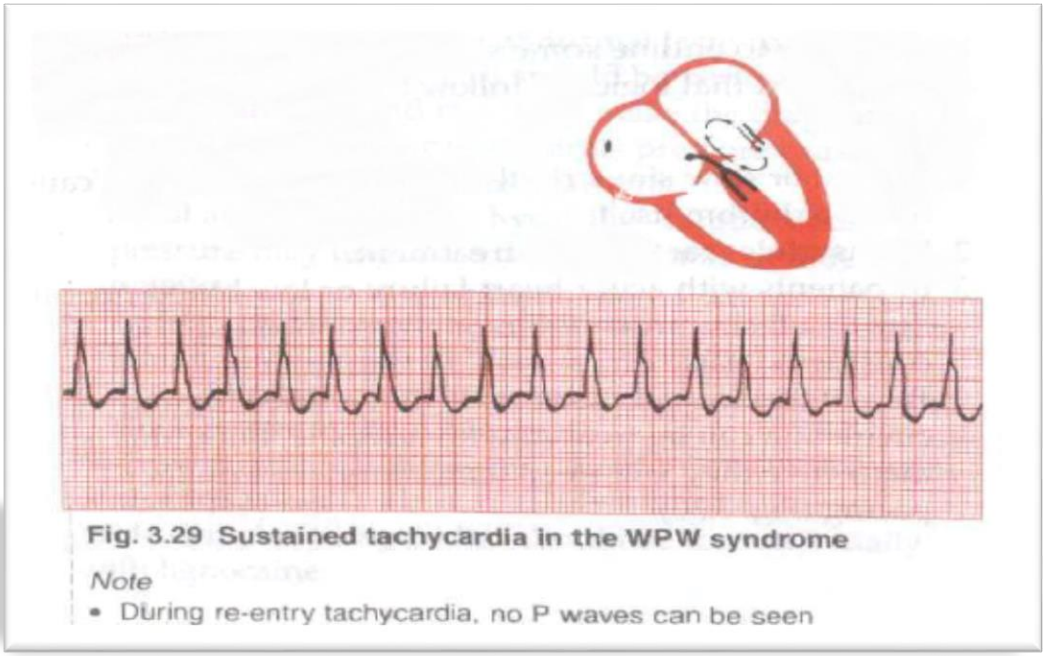


Fig. 3.29 Sustained tachycardia in the WPW syndrome

Note

- During re-entry tachycardia, no P waves can be seen

Abnormalities

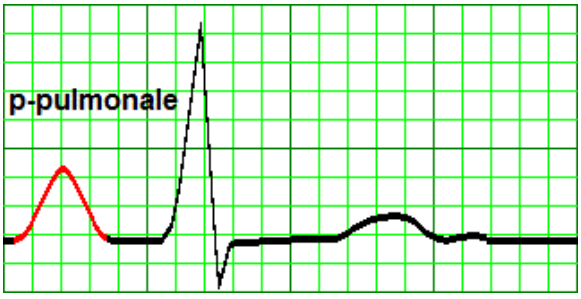
1) Abnormal P-wave:

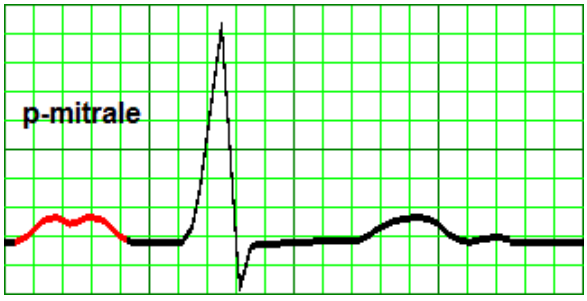
1. P-pulmonale (peaked "tall & narrow" p-wave):
   in Rt. Atrial abnormalities
   e.g.: tricuspid stenosis, pulmonary HTN
2. P-mitrale (broad & bifid) :
   in Lt. Atrial abnormalities
   e.g.: mitral stenosis

Normal P-wave:

- Amplitude = 2.5 mm
  = 2.5 مربعات صغيرة
- width = 3 mm
  = 3 مربعات صغيرة

N.B: Tall and wide P-wave = combination of Lt. & Rt. Atrial abnormalities





2) Abnormal QRS complex:

Normal QRS

1. **Duration (width):** ≤120 ms 3مربعات صغيرة
2. **Height:** ≤25 mm 6-5 مربعات صغيرة
3. **In V1 :** S-wave > R-wave
4. **In V6 :** R-wave > S-wave
5. **Q-wave** may be present in Lt. ventricle leads dt. Septal depolarization. But, these are < 1 small square width & < 2 small sruares depth.

Abnormal width: 1. BBB
2. Vent. Rhythms:

↑↑
- vent. Escape
- vent. Extrasystole
- vent. Tachycardia

Abnormal height:

↑↑ when ventricular muscle mass increase dt. Pressure or volume overload.

It may ↑↑ in thin people which is completely NORMAL.

Origin of Q-wave:

- In Lt. ventricular leads (V5,V6), small Q-wave result from depolarization of septum from Lt. to Rt.
- Normally: 1mm width & < 2mm depth.
- If Q-wave > normal → MI

3) Abnormalities of ST segment :

Normally , iso-electric "same level between T-wave & next P-wave"

J-point: the junction between end of QRS complex & beginning of ST segment.

Elevated ST segment	Depressed ST segment
J-point above iso-electric line.	J-point above iso-electric line.
1. Acute "early" ischemia & MI	1. Late ischemia & MI
2. Pericarditis: diffuse ST elevation	2. Digoxin toxicity
3. Prinzmetal's angina: transient.	3. Hypokalemia
4. Acute myocarditis.	4. May be with LVH
5. May be LVH & LBBB: in V1,V2	5. Exercise (normal)

ST segment changes

ST segment elevation in MI:

1. **Ant. Wall MI (ant. & lat. Of Lt. ventricle):**
   Leads I & aVL & V1 – V6
2. **Post. Infarction:**
   V7, V8, V9
3. **Rt. Infarction:**
   V3R, V4R
4. **Inf. Wall infarction:**
   II, III, aVF

ملحوظة:
Elevation = 1 small square in all leads
EXCEPT V1, V2, V3. ➔ males مربعين
↓
Females مربع ونصف

- One of the most important characteristics of ST – T change is their reciprocity.
- Ant. & inf. Leads tend to show inverse pattern.

4) Abnormalities of T-wave:

Inverted T-wave is seen in :

1. **Normality (Mostly in V1).**
2. **Ischemia "late".**
3. **Ventricular overload.**
4. **BBB.**
5. **Digoxin toxicity.**

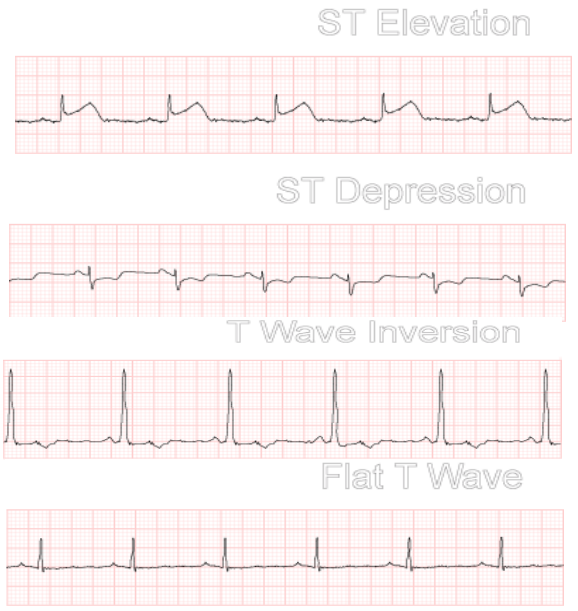
1. Normality:

T-wave may be inverted in young or Black people.

2. Ischemia & MI :

Changes in Order :

1. ST elevation.
2. Q-wave appears.
3. Normalization of ST segment or even depression.
4. Inverted T-wave.
5. Flat T-wave.



3. Ventricular overload:

Rt. Ventricular hypertrophy	Lt. ventricular hypertrophy
• Best seen in V_1 (Rt. Vent. Lead) • Tall R-wave in V_1 >25 mm. • Deep S-wave in V_6 • May be: - RBBB - Rt. Axis deviation - Inverted T-wave in V_1	• Best seen in V_5,V_6 (Lt. Vent. Lead) • Tall R-wave in V_5,v_6 >25 mm. • Deep S-wave in V_1 • May be: - Lt. Axis deviation - Inverted T-wave in V_5,V_6

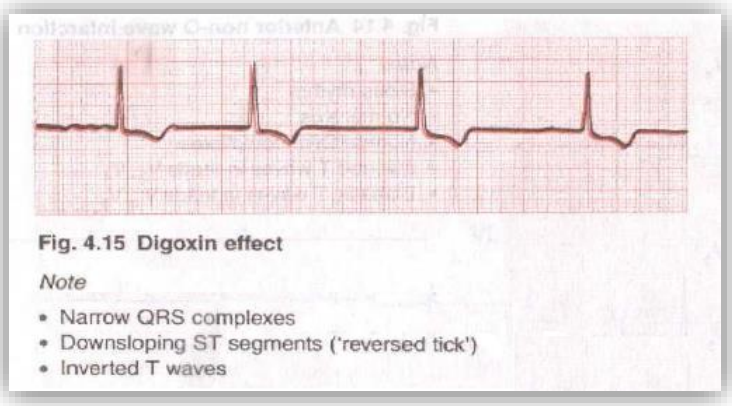
N.B: In Lt. vent. Hypertrophy: depth of S-wave in V1 + height of R in V6 > **35** mm

4. BBB:

The abnormal path. Of depolarization in BBB usually associated with abnormal path. Of repolarization. So, may we see inverted T-wave associated with QRS≥ 160 ms.

5. Digoxin toxicity:

- St segment depression "downsloping" شكلها مميز
- inverted T-wave.



5) Abnormalities of ST segment & T-wave in Electrolytes disturbance: (الصوديوم مابيعملش تغييرات في رسم القلب)

↓ K or ↓ Mg : Flat T-wave & appearance of U-wave "hump on end of T-wave"

↑ K or ↑Mg : - peaked T-wave & disappearance of ST segment.

- QRS may widened

↓ Ca : ↑ QT interval

↑ Ca : ↓ QT interval علاقة عكسية

تم بحمد الله